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Factsheet

ASCITES IN MEAT-TYPE CHICKENS CAUSED BY RIGHT HEART FAILURE

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Definition Ascites is an accumulation of non-inflammatory fluid in one or more of the abdominal spaces. There may be clots of yellow material in the fluid.

INTRODUCTION

Ascites caused by right heart failure (RHF) has been an important cause of illness and death in broiler chickens raised at high altitude for many years. The number of cases of ascites caused by RHF in broiler and roaster chickens in the U.K., Canada, the United States, and other areas of the world where broilers are grown at low altitude has increased over the past several years and coincides with a continuing genetic and nutritional improvement in feed efficiency and rate of growth. The incidence of ascites is above 1% in some broiler flocks and many roaster flocks, and it is occasionally 15 to 20% in roaster flocks.

Increased pulmonary (lung) arterial pressure can be produced in mammals and birds by lack of oxygen. The heart must circulate blood more rapidly to provide the same amount of oxygen to the body. In both chickens and mammals lack of oxygen in the body causes a marked increase in the number of blood cells, and this makes the blood more viscid and more difficult to pump through the lung. The right side of the heart enlarges in response to the increased work and this eventually leads to RHF if the heart has to continue working harder than normal.

At low altitude it is not lack of oxygen in the air that causes the increased work for the heart and dust or fumes in the pens are not likely causes unless they cause pneumonia. Carbon monoxide interferes with oxygen uptake and carbon monoxide levels (from the brooders) have been above normal in some barns where RHF has been a problem. The most important cause of increased work for the heart at low altitude appears to be the high oxygen requirement of rapid growth in the modern broiler combined with restricted space for blood flow through the small blood vessels of the lung.

CLINICAL SIGNS

Occasionally young broilers will develop ascites, particularly if there is too much salt in the feed or water of if lung diseases like aspergillosis are present, but if the lung is normal, deaths from ascites is greatest after five weeks. Clinically affected broilers have a pale head and a shrunken comb and in white chickens the feathers lose their bright white sheen. The abdominal skin may be red and vessels in the skin congested. Since growth stops as RHF develops, affected broilers are smaller than their pen mates. Their abdomen is distended with fluid and the pressure results in an increased respiratory rate and reduced exercise tolerance.

POSTMORTEM EXAMINATION AND DIAGNOSIS

At postmortem there is a large or small quantity of clear yellow fluid and clots of fibrin in the abdomen. The liver may be swollen and congested or firm and irregular with edema (fluid), and have fibrin adherent to the surface. It may be nodular or shrunken, it may be white with edema under the capsule or have large or small blebs of edema in the sacs around the liver. There is a mild to marked increase in fluid in the sac around the heart and occasionally there is inflammation of the outer surface of the heart. There is enlargement of the right side of the heart and thickening of the muscle of the right side. The right atrium and vena cava are very dilated. Frequently there is thinning of the left heart. The lungs are extremely congested and edematous.

Not all broilers which die from RHF have ascites. Death may occur before clinical signs are observed and affected broilers frequently die on their back. At necropsy there may be a swollen liver, venous congestion, a dilated right atrium and vena cava and thickening of the right heart, as well as marked lung congestion and edema (death is likely from respiratory failure). The intestine may or may not be empty but the heart changes will differentiate RHF from flip-over.

WHY DO MEAT-TYPE CHICKENS GET ASCITES?

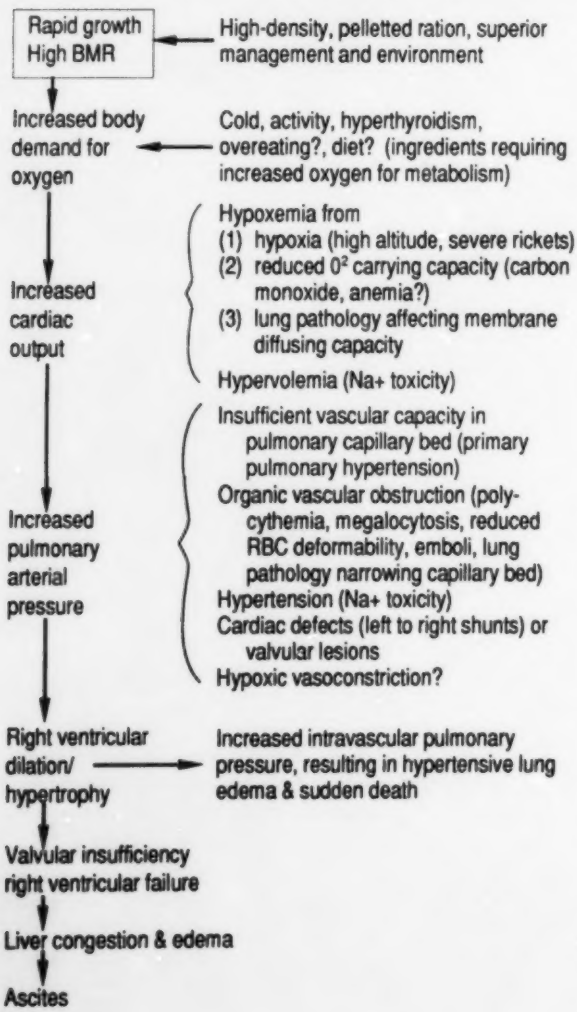
The lungs of birds are rigid and molded into the thoracic cavity. They cannot expand like mammalian lungs. The capillaries can expand only very little to allow for increased blood flow. The lungs of chickens grow less rapidly that the rest of the body, and lung capacity does not keep up to the very rapid growth of muscle in fast growing broiler chickens.

In birds the right side of the heart is very thin walled and is meant to be a volume, not a pressure pump. When increased pressure occurs, the heart responds to the increased workload, as muscle does, by getting bigger. If pressure remains high, the right heart has to pump against that pressure and the muscle continues to thicken and get larger until the valve will no longer cover the opening back to the body, then valvular insufficiency and RHF occur. Blood pressure rises in the vena cava resulting in increased pressure in the liver, leakage of fluid from the liver and ascites.

Ascites caused by RHF has been produced by rickets which makes breathing difficult and increases the number of cells in the blood. Cold is a common cause of outbreaks of ascites in small farm flocks because it increased the blood flow through the lung. It would appear that ascites at low altitude may be caused by lack of sufficient space for blood flow in the lung and that predisposing factors which increase the amount of oxygen required, reduce oxygen carrying capacity of the blood, increase blood volume, or interfere with blood flow through the lung may result in flock outbreaks of ascites. A diagrammatic summary of possible causes of increased pulmonary arterial pressure is shown in Figure 1.

Figure 1.

DIAGRAMMATIC SUMMARY OF POSSIBLE CAUSES OF INCREASED PULMONARY ARTERIAL PRESSURE IN BROILER CHICKENS RESULTING IN INCREASED RIGHT VENTRICULAR WORK-LOAD


Rapid growth
High BMR
High-density, pelletted ration, superior management and environment
Increased body demand for oxygen
Cold, activity, hyperthyroidism, overeating?, diet? (ingredients requiring increased oxygen for metabolism)
Increased cardiac output
Hypoxemia from
(1) hypoxia (high altitude, severe rickets)
(2) reduced 0^2 carrying capacity (carbon monoxide, anemia?)
(3) lung pathology affecting membrane diffusing capacity
Hypervolemia (Na+ toxicity)
Increased pulmonary arterial pressure
Insufficient vascular capacity in pulmonary capillary bed (primary pulmonary hypertension)
Organic vascular obstruction (polycythemia, megalocytosis, reduced RBC deformability, emboli, lung pathology narrowing capillary bed)
Hypertension (Na+ toxicity)
Cardiac defects (left to right shunts) or valvular lesions
Hypoxic vasoconstriction?
Right ventricular dilation/ hypertrophy
Increased intravascular pulmonary pressure, resulting in hypertensive lung edema & sudden death
Valvular insufficiency right ventricular failure
Liver congestion & edema
Ascites

Adapted from:
Julian, R.J. The effect of increased sodium in the drinking water on right ventricular hypertrophy, right ventricular failure and ascites in broiler chickens. Avian Pathology 16: 61-71, 1987.

CONTROL

Ascites caused by primary pulmonary hypertension can be prevented by slowing growth to reduce the oxygen requirement after 30 to 35 days of age. This can be done by restricting feed, feeding a mash diet, or using a less dense diet. Long dark periods are also effective but should be started at day 3 and require covered fan outlets. Limiting the number of hours of light will slow growth and will reduce the incidence of flip-over and lameness and leg deformity as well as ascites. A lighting program of 3 hours light to nine hours dark may be effective. Each light period should be lengthened by 1 hour a week starting at day 18. Environmental temperature, humidity and air movement should be controlled to prevent excessive body heat loss. Ascites caused by other factors can be prevented by avoiding the cause. Moldy litter must be avoided. Sodium level in feed should not be above 2000 parts/10^6. Feed should be routinely analyses for sodium directly not estimated from the chloride level since high levels of sodium from sources other than NaCl may be present. If sodium is present in the water supply, sodium in the feed should be reduced by 2 to 3 times the level present in the water. (For 500 parts/10^6 of sodium in the water reduce feed sodium 1000-1500 parts/10^6). Water containing over 1000 parts/10^6 of sodium should not be used for young broilers. Altitudes above 1800 to 2000 m are unsatisfactory for meat-type chickens and growth must be slowed to prevent mortality. More care to prevent chilling is also necessary at high altitude.

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